



UNITED STATES NAVY *Medical News Letter*

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Surgeons General of the Past

(The third in a series of brief biographies)

The third Chief of the Bureau of Medicine and Surgery, William Whelan, was born in Pennsylvania on 4 September 1808, graduated from the University of Pennsylvania Medical School in 1828, and was commissioned a surgeon's mate in the Navy on 25 March of that year. His military service included duty with the West Indian Squadron, at the naval hospital in Pensacola during a high incidence of smallpox and yellow fever, at the Boston Naval Hospital and the Philadelphia Navy Yard. Dr. Whelan was Fleet Surgeon of the Mediterranean Squadron from 1843-1845 and 1849 to 1852 and also served aboard the USS Falmouth in the Pacific. Appointed Chief of Bureau in 1853 he served until 1865, the longest tenure of anyone who has occupied this high position in the Navy, and thus directed the medical service during the Civil War. Under his leadership new hospitals were commissioned at Annapolis in 1853 and at Washington, D.C. in 1861; and the RED ROVER on 26 December 1862 became the first American naval vessel commissioned as a hospital ship. She also had aboard the Navy's first women nurses, the Volunteer Nursing Sisters. The war caused 3,266 gunshot and powder wounds to naval personnel. Disease casualties were more numerous. The use of the hypodermic syringe and improved anesthesia occurred during this period; and Surgeon G. R. B. Horner of the Gulf Squadron wrote a valuable pioneering book on the "Diseases and Injuries of Seamen" for the guidance of the 200 physicians in the Navy's Civil War Medical Department. Dr. Whelan's term ended with his death in office on 11 June 1865.

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MEDICAL NEWS LETTER

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DRESSLER SYNDROME (POSTMYOCARDIAL INFARCTION SYNDROME)

*Emanuel J. Levin MD and David Bryk MD, Radiology 87(4): 731-736,
October 1966.*

In 1956, Dressler described a hitherto unreported syndrome arising as a sequel or complication of myocardial infarction. Many of these cases formerly had been considered examples of pulmonary infarction, recurrent myocardial infarction, or extension of myocardial infarction. Although proof is not available, it is believed that the Dressler syndrome is an expression of an autoimmune reaction and is similar to or identical with the group of post-cardiotomy syndromes, the traumatic pericarditis syndrome, and the idiopathic benign pericarditis syndrome.

More than 100 cases of the post-myocardial infarction syndrome have been available for clinical studies at Maimonides Hospital, and of this group 52 have had adequate radiographic examinations. In many instances, the roentgen examination has supported or corroborated the clinical impression of the syndrome, and at other times it has suggested the diagnosis when it was not under clinical consideration.

The purpose of this report is to present the roentgen findings in these 52 patients with the Dressler syndrome, and at the same time summarize its pertinent clinical elements.

General Considerations

The essential elements of the syndrome, pericarditis, pleuritis, and pneumonitis, are usually present in combination, but each may occur as a sole manifestation. The illness may be ushered in within a few days of the onset of the myocardial infarction or may be delayed as long as eight weeks. Pain of the pleuropericardial type is typical, together with low-grade fever, leukocytosis, and elevation of the sedimentation rate. A pericardial friction rub is found in about 80 percent of the cases and is more persistent than the transient rub of pericarditis epistemiocardiaca. The tendency toward relapse is a conspicuous feature of the syndrome,

and there may be as many as six episodes, each lasting from one to six weeks.

The prognosis is usually excellent, and the illness pursues a benign course. Even when the symptomatology is severe, dramatic improvement can be expected with the institution of steroid therapy. In rare instances, however, there may be severe intensification of the disease process and death.

Postmortem examinations were obtained in three of these patients: in two during the course of the post-myocardial infarction syndrome and in the third one year subsequent to the initiating infarct and in the course of a second infarct. In the first two cases, there were 700 and 380 ml of hemorrhagic pericardial fluid, respectively, and the visceral and parietal pericardium appeared shaggy and covered with thick granular fibrinous tissue. The infarcts in one case were in the posteroseptal myocardium and in the other in the anteroseptal wall. They were three to five weeks old. In the third case, there was no effusion, but the pericardial cavity was obliterated by chronic adhesive pericarditis, indicating previous pericardial disease, presumably that related to the episode one year earlier. In this patient healed infarcts were observed in the posterior and anteroseptal areas, and there was a recent infarct in the posterolateral wall of the left ventricle.

In the two patients who died during the course of the syndrome pleural effusions were present: 600 ml of clear fluid in each pleural space in one case, and 1,000 ml and 150 ml of hemorrhagic fluid in the left and right pleural spaces in the other. The first case exhibited severe congestive changes and minimal basilar atelectasis, and the second, moderate atelectasis and nonspecific interstitial pneumonitis.

In the patient with pleural and pericardial hemorrhagic fluid, death appeared to be related to the use of anticoagulants. Since anticoagulant therapy is generally considered to be contraindicated in the Dressler syndrome but may be acceptable in the treatment of myocardial or pulmonary infarction, it

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is essential that the correct diagnosis be made expeditiously.

Roentgen Findings

In 25 of the 52 cases studied, there was roentgen evidence of all three manifestations of the syndrome: pericardial effusion, pleural effusion, and pneumonitis. In 22, pleural effusion and/or pneumonitis existed alone or in combination, but there were no cases of pericardial effusion unassociated with either pleural effusion or pneumonitis. Five roentgen studies were completely negative despite clinical evidence of the syndrome.

1. Cardiovascular Silhouette

A rapid increase in the size of the cardiac shadow, particularly in the absence of congestive heart failure, is one of the more reliable signs of pericardial effusion. This finding was observed in 36 of the 47 cases in which films were available for study both during and before or after the syndrome. Only bedside or supine films could be obtained during the severe phases of the illness in many instances, so that at times gross comparisons alone could be made. In 29 patients there was pre-existing cardiac enlargement due to hypertensive arteriosclerotic heart disease.

On the lateral projection, when available, the pericardial effusion could usually be detected by a decrease in the expected size of the retrosternal air space or an unusual bulge of the anterior surface of the cardiac shadow. This appearance is explained by the tendency for this effusion to accumulate anteriorly.

In 17 cases the pericardial effusion caused a decrease or even a loss of the concavity normally present in the left heart border. This straightening of the left cardiac contour was accentuated in those patients with left ventricular enlargement in whom the concavity is usually prominent.

Widening of the superior mediastinum was apparent in 20 cases. It was the result of two distinct processes, but their simultaneous occurrence was frequent.

(a) Widening to the right, occasionally with lateral convexity, was due to engorgement of the superior vena cava and the right innominate vein. This finding correlated with increased venous pressure and effected the increased tension in the pericardial sac.

(b) Widening of the superior mediastinum to the left was the result of distention of the superior pericardial recess. In some cases, the lateral border of the widening recess overlapped the contours of

the aortic knob and its outlines were obliterated on underpenetrated films. In films of sufficient penetration, however, the superimposed shadows of the aortic knob and the distended pericardial recess could be distinguished.

Two cases showed bilateral mediastinal widening with ill-defined lateral borders associated with obscuration of the aortic knob on properly exposed films. In one, the aortic knob was calcified and the distance between this calcification and the adjacent aerated lung was increased. In view of the posterior localization of these changes, they could not be attributed to pericardial disease, but more than likely were caused by inflammatory processes in the adjacent mediastinum, pleura, or lung.

Pericardiocentesis was rarely required as a therapeutic procedure, particularly since rapid improvement usually followed the administration of steroids. In the few cases in which air was injected into the pericardial sac following tap, the pericardium was thick and shaggy.

2. Pleura

Pleural effusions were present in 46 cases. They were bilateral in 22, and in 15 of these were greater in extent on the left. Unilateral effusions in 24 cases were equally divided between the two sides. Usually the fluid amounted to little more than blunting of the costophrenic sinuses, but in a few cases it was sufficient to cause respiratory embarrassment, and thoracenteses were required. In 18 patients, the fissures were widened by interlobar fluid but only to a minimal degree.

The severity of the pleural effusions roughly paralleled that of the pericarditis and pneumonitis, but the effusions tended to resolve more slowly.

In a few cases, the descending aorta was not visualized in a properly penetrated frontal projection. In its place an indistinctly outlined, irregular infiltrate was seen. Although it is probable that pleural disease accounted for the loss of outline of the aorta, it is also possible that mediastinal or pulmonary disease was the cause.

3. Lungs

There were pulmonary infiltrates in 32 cases. In 12 they were bilateral, and in 14 restricted to the left lung. The bases of both lungs as well as the middle lobe and lingula were involved usually, but the upper halves rarely and then only minimally. The roentgenographic patterns demonstrated were variations of nonhomogeneous, patchy, ill-defined

areas of infiltrate, interspersed with zones of segmental and subsegmental atelectasis.

During the active phase of the syndrome, the pulmonary vasculature was normal in 40 patients. In a few venous congestion was consistent with left heart failure of a mild degree. Pulmonary edema was present occasionally, but was not seen as a manifestation of the postmyocardial infarction syndrome and represented another manifestation of myocardial damage.

Differential Diagnosis

The postmyocardial infarction syndrome may be confused with pulmonary embolization or infarction, another myocardial infarction, or extension of previous myocardial infarction. None of these conditions, however, cause prolonged or relapsing episodes of fever, pericarditis, pleuritis, and pneumonitis. Furthermore, in the postmyocardial infarction syndrome, steroid therapy is followed by a dramatic alleviation of signs and symptoms, with frequent relapses upon its withdrawal.

The sudden onset of pleural pain, hemoptysis, and dyspnea, often with signs of collapse and electrocardiographic evidence of acute cor pulmonale without a pericardial rub, supports the diagnosis of a pulmonary infarct.

In uncomplicated fresh myocardial infarction or extension of a myocardial infarct, there is no pleural or pericardial effusion nor is there pneumonitis. A pericardial friction rub, when present, is transient. Lastly, the electrocardiographic patterns of myocardial infarction are usually seen and are diagnostic.

Roentgenograms in about three-quarters of the cases will indicate the presence of pericardial

effusion. Widening of the superior mediastinum to the left, due to distention of the superior pericardial recess, and to the right, from engorgement of the superior vena cava and innominate vein, appears to be a rather characteristic roentgen pattern in about a third of the cases. Erect, postero-anterior roentgenograms, however, must be obtained, even at the bedside, since the mediastinum normally appears wider on supine anteroposterior films, thus simulating the abnormal configuration seen in the syndrome. The associated bilateral or unilateral pulmonary infiltrates and pleural effusions are generally nonspecific in appearance, but in association with the roentgen cardiac findings they frequently suggest the diagnosis.

Summary

The postmyocardial infarction syndrome is characterized by the occurrence of pericarditis, pleuritis, and pneumonitis a few days or weeks subsequent to a myocardial infarction. Its symptomatology resembles that of a myocardial or pulmonary infarct. The roentgenograms usually demonstrate rapid increase in the size of the cardiac shadow and other features of pericardial effusion in association with pulmonary infiltrates and pleural fluid. Attention is called to a characteristic widening of the superior mediastinum which is seen in about one third of the cases.

It is essential that the correct diagnosis be established because anticoagulant therapy, which is acceptable in the treatment of infarct, is contraindicated in the postmyocardial infarction syndrome.

(The references and figures may be seen in the original article.)

TRAUMATIC DISLOCATION OF THE INCUS ASSOCIATED WITH BASILAR SKULL FRACTURE

A TREATABLE CAUSE OF DEAFNESS FOLLOWING CRANIAL TRAUMA*

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Department of Neurological Surgery and Department of Otolaryngology,
U. S. Naval Hospital, Charleston, South Carolina. J Neurosurg
24(2): 570-572, February 1966.*

A neurosurgeon is frequently presented with the problem of a patient with hearing loss following head injury. This head injury is often associated with a skull fracture, particularly one involving the temporal bone. All too often, the resultant hearing loss is not adequately investigated. This paper presents a case in which the hearing deficit following cranial trauma was due to a traumatic dislocation of the incus which was corrected by transposition of the malleus to the head of the stapes providing an intact middle ear sound conduction mechanism.

Case Report

A 19-year-old sailor was admitted to the neurosurgical service of the U.S. Naval Hospital, Charleston, South Carolina, on October 5, 1964, following an automobile accident. He had been riding in the front seat of an automobile which was involved in a head-on collision. He was immediately rendered unconscious.

Examination. On examination, the patient was comatose, with a blood pressure of 150/90, pulse 60, respirations 20. There were bilateral contusions of the scalp in the frontal area, a large subgaleal hematoma underlying the left frontal scalp, and multiple puncture wounds due to glass in the frontal scalp. The pupils were equal and reacted to light. The fundoscopic picture was normal; in particular, there were no hemorrhages and no papilledema. Examination of the ears revealed a cerebrospinal fluid otorrhea on the right, and blood behind the left tympanic membrane. There was a fracture of the right clavicle. The chest, abdomen and extremities were normal.

Admission skull x-rays showed a linear fracture of the right parietal bone. In addition, there were bilateral linear fractures of the temporal bones descending into the base of the skull. Mastoid films

revealed a questionable defect in the tegmen tympani adjacent to the epitympanic area; this probably was part of the basilar fracture and thus related to the traumatic displacement of the incus discovered later. Chest x-rays revealed a comminuted fracture of the right clavicle. The lung fields were clear. The remaining laboratory examinations were normal except for a gross hematuria which cleared on the day of admission. The patient was treated in the intensive therapy ward and a figure-of-eight dressing applied for the right clavicular fracture. Prophylactic antibiotics were initiated. His state of consciousness rapidly improved but he continued to have a persistent cerebrospinal fluid otorrhea on the right side. The right ear was covered with a sterile gauze square which was changed every 2 hours. Early on the evening of admission, a small spicule of bone mixed with the blood and cerebrospinal fluid was noticed in the sterile dressing. Closer examination of this revealed it to be the incus (Fig. 1). Later that evening a small amount of brain tissue exuded from the right ear canal. A lumbar puncture on the day following admission revealed a pressure of 480 mm. The spinal fluid contained 765 red blood cells and a total protein of 149 mg percent. The cerebrospinal fluid pressure was decreased by multiple lumbar punctures and the use of intravenous urea. With this treatment, the spinal fluid pressures were kept in the 120-140 range and there was no further extrusion of brain tissue from the right ear. The patient continued to make an uncomplicated recovery and 3 weeks after his accident his only complaints were occasional headaches and a right-sided hearing loss. There was good strength in all of his extremities and no evidence of facial nerve paresis or vestibular disorder.

On October 23, 1964, he was placed in a plaster jacket for further immobilization of the clavicular fracture. Audiometric evaluation at this time revealed a significant conduction deafness in the right ear. He was therefore transferred to the otolaryn-

*The opinions or assertions contained herein are the private ones of the author and are not to be construed as official or as necessarily reflecting the views of the Department of the Navy or the Naval Service at large.

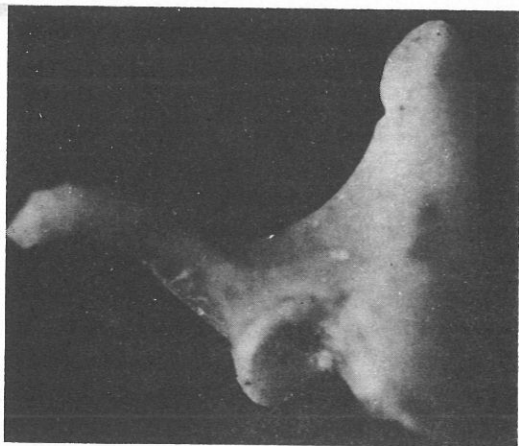


Fig. 1. Photograph of incus which spontaneously extruded from the right ear canal following a basilar skull fracture.

gology service for operative repair of the break in continuity of the ossicular chain. Otolaryngological evaluation in November, 1964, revealed the right external ear canal to be normal in size and shape. The right tympanic membrane contained a central tympanosclerotic plaque with some scarring posteriorly and superiorly. The Eustachian tube was open to self inflation and there was no evidence of fluid in the middle ear. Tuning fork tests suggested a conductive type of hearing loss on the right

and lateralization of the Weber to the right. The left tympanic membrane and ear canal were normal. The nasopharynx was clear and the Eustachian tube orifice on the right did not reveal any drainage of fluid that would suggest a cerebrospinal fluid leak into the nasopharynx.

Operation. On March 23, 1965, exploration of the right middle ear was undertaken under local anesthesia, utilizing the Zeiss operating microscope. There was marked scarring and fibrosis of the middle ear space. Fibrous tissue completely enveloped the superstructure of the stapes. Inspection of the head of the malleus and the epitympanic area resulted in a cerebrospinal fluid leak that was readily controlled with pressure packing in the middle ear. The sound conduction mechanism was reconstructed by freeing the head of the malleus from its ligaments and adhesions as well as transecting the tensor tympani muscle and then rotating the head of the malleus onto the head of the stapes.

The postoperative course was uneventful. There was no evidence of spinal fluid drainage. Ten days following surgery, examination indicated that air conduction was greater than bone conduction with a 500 cycle per second fork. During the next 4 weeks the patient's hearing continued to improve and audiograms upon discharge were as in Table 1. The patient returned to full duty on April 22, 1965.

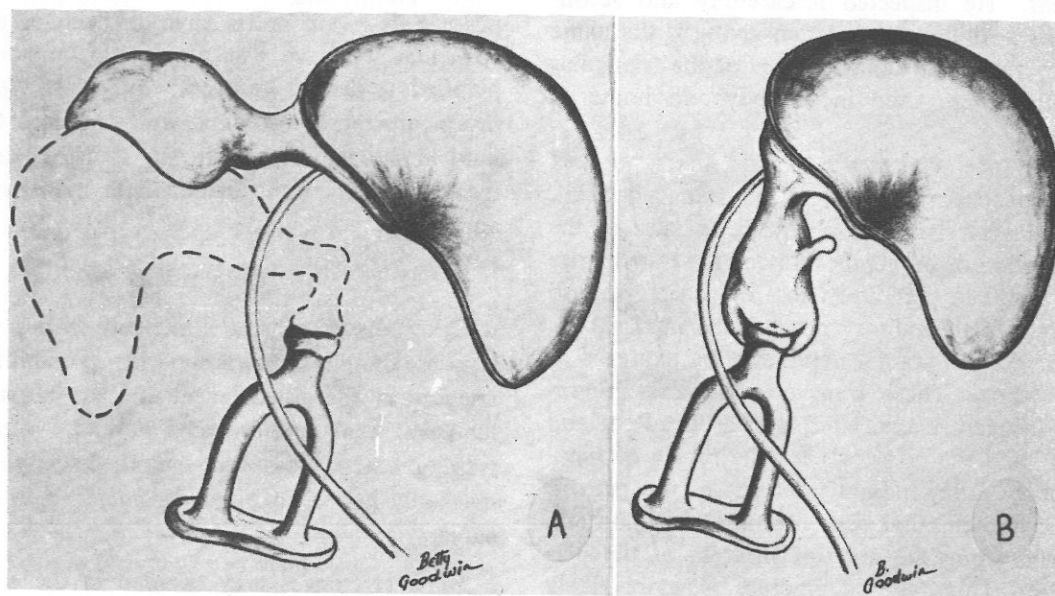


Fig. 2. (a). Artist's view through the Zeiss operating microscope showing absence of the right incus (dotted line). (b). Postoperative view showing the articular surface of the head of the malleus transposed onto the head of the stapes, thus providing an intact middle ear sound conduction mechanism.

TABLE 1
Audiograms

Cycles per Second	250	500	1000	2000	4000	8000
Three weeks after accident (loss in decibels)	50	55	55	35	60	60
Four weeks after operation (loss in decibels)	5	5	10	15	20	20

Discussion

It is interesting that the presence of ossicles within the inner ear escaped the notice of such physicians as Democritus, Celsus and even Galen. It was left for the Italian physician, Bengario da Carpi, to give the first recorded description of the malleus and incus, describing them as "two small ossicles adjacent to the aforesaid panniculus (tympanic membrane) which are moved by the moving ear and strike one against the other." Da Carpi not only described the ossicles but attempted to explain their function by their striking together much as a hammer strikes an anvil. It remained, however, for Vesalius to point out the anatomical similarity of the 2 ossicles to a hammer and anvil and hence to name them the malleus and the incus.

A quarter of a century was to lapse before the third ossicle in the ossicular chain was discovered and described by the Sicilian physician, Gian Filippo Ingrassia. In this instance, chance certainly favored the prepared mind, for while preparing a skull for anatomical demonstration, Ingrassia noted that a small ossicle had fallen to the surface of the table as he was cleansing the middle ear. He inspected it carefully and reconstructed its position and function giving it the name of stirrup (stapha) in remembrance of the triangular stirrups that were used in his boyhood home of Sicily.

How may one suspect a traumatic interruption of the ossicular chain following head trauma? Schiff has stated that transverse fractures across the petrous portion of the temporal bone are much more likely to result in permanent loss of hearing due to transection of the auditory nerve. The intrapetrous portion of the 7th cranial nerve may be damaged at the same time. These transverse fractures, therefore, are frequently associated with a "dead ear and labyrinth" and the prognosis for the return of function is poor. Longitudinal fractures of the petrous portion of the temporal bone are more common and usually run along the anterior buttress of the petrous ridge. This type of fracture is more likely to cause disturbance in the middle ear leading to a

conductive type of hearing loss with essentially normal vestibular function, a type of lesion that is readily accessible to the otologist and may be corrected surgically. Kossner has stated that a unilateral conductive type of hearing loss with a marked air-bone gap of up to 60 decibels and with an intact tympanic membrane should arouse one's suspicion that there has been an interruption of the ossicular chain. A conduction type of deafness after head injury with a fracture of the temporal bone or an extruded portion of ossicle should suggest the diagnosis.

The importance of the incus in the ossicular chain is underscored by Gundersen. He states that in chronic otitis media it is usually the long process of the incus which is destroyed first. He found that in 190 ears operated on for chronic otitis media at the Haukland Hospital, the long process of the incus was destroyed in 74.2 percent (141 ears). The long process of the incus is the weak link in the ossicular chain, not only in infection, but also in trauma.

Various techniques for repair of the ossicular chain have been described. Polyethylene struts, wire-veins attached to the malleus handle, fat plugs, and repositioning of the deficient incus, Teflon pistons, and vein grafts have all been used. In the case that we have just presented, we had initially planned to use a notched, clothespin type Teflon piston prosthesis but when we discovered the spinal fluid in the middle ear, it was thought best to perform a malleal transposition with malleus-stapedial apposition.

Conclusions

Clinicians in charge of patients with severe head injuries should be aware of the possibility of disruption in the ossicular chain. A patient with a temporal bone fracture who develops a conduction type of hearing loss may have a defect that can be markedly improved by replacement or transposition surgery.

(The references may be seen in the original article.)

HOW NOT TO TREAT ANEMIA

John P. Mahoney MD, Med Clin N Amer 50(6): 1713-1720, November 1966.*

The purpose of this article is to call the attention of clinicians to some widespread misconceptions about anemia and some widespread practices in the management of anemic patients which deserve condemnation.

When I was asked to prepare this article, I argued that another paper on the subject was unjustified since so many have already been written. "The empirical approach to the patient with anemia has been under condemnation for 30 years," I said. "No one in 1966 would treat a patient without first making a diagnosis, and no one would treat a patient with combinations of drugs when a specific remedy is available." Shortly after our conversation, the following patient was referred to me. This patient illustrated so many errors in management that I decided a discussion of how not to treat anemia would, indeed, be appropriate.

Case Report

A 33 year old unmarried woman, a teacher, was referred because of anemia of 9.5 gm/100 ml. She had nonspecific complaints of mild fatigue at the end of her working day, general lack of energy, and scanty menstrual flow. She felt that her complexion was sallow. She had no other symptoms.

Past history revealed that she had been anemic at age 16 and had been treated with iron. One year previously she had had a nasal polypectomy under general anesthesia and had been given 2 units of whole blood prior to the operation. There was no history of exposure to toxic chemicals, and she took no medications except those mentioned above. Her mother and father were of Portuguese ancestry. Both were dead. She had six siblings, one of whom had been found to be anemic recently and had consulted another Boston hematologist. No diagnosis had yet been made, however, and no therapy had been prescribed. The patient had stated that her hemoglobin concentration was 9.7 gm/100 ml. She received ferrous gluconate, 2 tablets daily for the past 9 months and received injections of vitamin B₁₂ twice weekly for the same period. She stated that she felt better after the injections and that under therapy her hemoglobin had risen to 10.2 gm. Be-

cause of failure of the hemoglobin to rise to normal levels, the patient was referred for evaluation.

Physical examination was entirely negative. There was no enlargement of the liver, spleen, or lymph nodes. The complexion was dark, but marked pallor was not present.

Blood values were as follows: RBC 4.70 m/cu mm, Hb 9.7 gm/100 ml, hematocrit 35 percent, reticulocyte count 2.1 percent, WBC 5100 per cu mm, polymorphonuclears 66, lymphocytes 26, monocytes 2, eosinophils 3, band cells 3. Red blood cells on smear revealed moderate hypochromia, moderate to marked poikilocytosis and anisocytosis, and about 2 percent of the cells were target shaped. There was a moderate degree of basophilic stippling.

Bone marrow was easily aspirated and showed a normal picture. Staining for iron by the Prussian blue reaction revealed excessive quantities of iron. Serum iron was 95 ug/100 ml. Iron-binding capacity was 300 ug/100 ml.

The patient's disease thus fell into the class of nonsideropenic hypochromic anemias and further study revealed an elevated A₂ hemoglobin and fetal hemoglobin of 6 percent. A diagnosis of thalassemia was thus established.

We advised the patient and her physician to discontinue therapy and counseled the patient that her mild symptoms were probably not due to the anemia, which was familial, had been present since birth, was untreatable, and had no serious implications for the patient.

Further investigation revealed that the patient's married sister had been seen by me 2 years previously. This fact was not known to the patient. A further distressing feature of this problem was that her sister had not been convinced that her anemia was not treatable and sought consultation with a second hematologist who confirmed our original impression of thalassemia minor. He also reechoed my statement that she did indeed have a familial, untreatable, but not particularly serious disorder and that her symptoms were probably not due to her disease.

I have taken the space to relate this case because I think it illustrated several points which must be made in any discussion of the treatment of anemia.

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Empirical Therapy

Erslev and McKenna, writing in this publication one year ago, stated, "The employment of empirical methods in the treatment of anemia is so widespread that patients (and many physicians) generally assume that good management of anemia consists of liver, vitamin B₁₂ injections, and iron. The success of this shotgun approach is undoubtedly caused both by the real need for iron by many patients and by the 'placebo effect' of medical attention and parenteral injections."

Our patient had received empirical treatment. It should not have been given for several reasons:

1. Anemia frequently is not treatable. Treatment with B₁₂ injections and iron in this case was a futile gesture, perhaps satisfying to the physician, but expensive, time-consuming, and useless to the patient. And perhaps not the least important, it was evidence of fuzzy thinking or perhaps lack of thinking on the part of the physician.

There are many methods of classifying anemias. I believe that one important classification that is often overlooked is the classification of patients into those whose anemia can and must be treated, and those for whom no treatment is available and who do not need to be treated.

2. It is not necessary to treat all anemic patients. Thalassemia minor is an example of a type of anemia for which there is no specific therapy. Only one method of therapy could possibly elevate the hemoglobin level in this patient: blood transfusion. But was it necessary to elevate the hemoglobin level? A vast experience with anemic patients indicates that they adjust quite well to mild degrees of anemia, although the concentration of hemoglobin at which individual patients have symptoms of fatigue, shortness of breath, and lassitude varies with each individual and certainly varies with the rate at which anemia develops. Patients with slowly progressing, chronic or congenital anemias may have no symptoms when their hemoglobin levels are greater than 8.0 gm per 100 ml and frequently are able to tolerate even lower levels.

This point is emphasized when one considers that we frequently make the diagnosis of thalassemia minor in a child who has had a routine pretonsillectomy hemoglobin which is below normal. When we make the diagnosis, we study siblings and parents. We often find that the father is anemic but has been performing hard physical labor all his life with no symptoms even though his hemoglobin is only 9.5 gm.

3. Not demonstrated by our case but needing consideration is the fact that treatment of anemia before diagnosis may obscure all subsequent attempts to make a diagnosis for many months. Nothing is more frustrating than to be called to see a patient admitted to the hospital with a fractured hip, found to have a hemoglobin level of 6.0 gm per 100 ml, transfused to 14.0 gm, and then as an afterthought, the hematology service invited to see the patient. We frequently have to recommend that such a patient return in eight weeks to be seen when we can examine her blood rather than the blood of the six donors.

4. Finally, patients should not be treated empirically because successful empirical treatment may lull the physician into a false sense of security that he has satisfactorily solved the patient's problem. In point of fact, the administration of iron to the iron-deficient patient may cause the hemoglobin to return to normal and will obscure the only clue to the early diagnosis of a gastrointestinal malignancy.

Our patient was fortunate. She had an anemia which did not respond to empirical "combination therapy." However, she might have had iron deficiency anemia due to blood loss from an early carcinoma of the cecum, and she might have responded to the administered iron, thus obscuring the only clue to the presence of a gastrointestinal malignancy. Too many patients present with symptoms of far advanced gastrointestinal malignancy who had obvious iron deficiency anemia successfully treated a year earlier.

Combination Therapies

The Physicians' Desk Reference lists 155 so-called hematinic preparations. A careful check reveals that most of these preparations contain not one but several materials. The companies which produce these materials do so for only one reason—they sell. They sell for only one reason—physicians prescribe them.

These facts deserve some consideration. First let us examine which anemias can be treated with hematinics. Hypochromic anemia due to iron deficiency needs to be treated with iron and nothing else. Megaloblastic anemia due to vitamin B₁₂ deficiency needs to be treated with vitamin B₁₂ and nothing else. The patient with megaloblastic anemia due to folic acid deficiency needs folic acid and nothing else. Most other anemias are either not treatable by specific substances or do not need to be treated. The presence on the market of preparations containing vitamin B₁₂, all the other vitamins, intrinsic factor, folic acid, iron, copper, molybdenum,

magnesium, manganese, ascorbic acid, zinc, etc., is a tribute to the salesmanship of the detail men who sell such compounds and the monumental gullibility of the American physician. It is not a tribute to the quality of medicine practiced in America in 1966.

The patient with megaloblastic anemia due to vitamin B₁₂ deficiency may have the true diagnosis masked by the administration of folic acid, and in fact, folic acid in large amounts may precipitate neurological complications of the disease.

Chronic administration of iron to patients with thalassemia or primary refractory anemia (sideroachrestic anemia) has the potential danger of causing iron overload with hemosiderosis and possible organ damage. One of the problems in these patients is their tendency to absorb abnormally large amounts of iron from the diet. They don't need to have this problem complicated by the administration of large quantities of therapeutic iron.

Slow-Release Iron Tablets (Spansules)

These preparations are sold with the following explanation of rationale: "Indicated for all simple iron deficiency anemias because the ferrous sulfate pellets in each capsule are widely dispersed for the gradual absorption of iron in the small intestine. Since few release their contents in the stomach, side effects are minimal. Because they produce virtually no gastrointestinal distress, they are ideal in pregnancy. For the same reason, they are particularly useful in patients intolerant to ordinary oral iron including convalescents, the elderly and patients with poor digestive processes, ulcer or gastrointestinal complaints." Left out of this statement is the abundant evidence that iron is absorbed in the upper gastrointestinal tract, and that administration of iron in a form deliberately designed to pass through the stomach and duodenum before being made available results in little or no absorption of the administered dose of iron. No absorption, no side effects.

Parenteral Iron

There is a widely held misconception that parenteral iron causes a more rapid production of hemoglobin than oral iron. The statement has been made that intravenous iron is indicated, for instance, in the anemic patient in the last trimester of pregnancy when there is insufficient time for hemoglobin regeneration by the oral route. There is no justification for such claims. The factor which limits the rate at which hemoglobin is regenerated is the ability of the bone marrow to produce hemoglobin, not the

amount of iron presented to the marrow. Indeed, most oral preparations of iron contain an excessive amount of the element which is responsible for the frequency of unpleasant side effects. Parenteral iron should never be given in an attempt to speed up the patient's response.

There are, of course, definite indications for parenteral iron therapy:

1. The patient who has a bleeding lesion which is not correctible, such as hereditary hemorrhagic telangiectasia. Here the patient may lose blood daily containing more iron than he can absorb. Since his bleeding lesion cannot be treated surgically, parenteral iron therapy may be the only solution to his problem.

2. Some patients may have bleeding gastrointestinal lesions which may be aggravated by oral iron. Ulcerative colitis and regional ileitis are examples of such lesions.

3. Patients who cannot be relied upon to take iron faithfully by mouth may be candidates for parenteral iron therapy.

4. Rarely a patient may be seen who complains of the side effects of oral iron and refuses to take the iron by mouth. If such a patient would rather remain anemic than take oral iron, such a patient might be a candidate for parenteral iron therapy.

In general, however, patients who are actually "intolerant" of oral iron include only approximately 6 percent of those taking 200 mg of iron daily. The problem of gastrointestinal intolerance has been needlessly exaggerated in an attempt to introduce newer compounds which are said to be better tolerated.

Transfusions

Andreas Libavius in 1615 wrote, "Let there be a young man, robust, full of spiritous blood and an old man, thin, emaciated, his strength exhausted, hardly able to retain his soul. Let the performer of the operation join the two—the hot and spiritous blood of the young man will pour into the old one as if from a fountain of life, and all his weakness will be dispelled."

Although modern blood banking techniques have made such an exchange possible, the results are not quite what Libavius expected. Unfortunately there are many physicians today who expect blood transfusions to accomplish the impossible.

Our patient received 2 units of whole blood prior to the performance of a relatively simple surgical operation. It is not clear what the surgeon was trying to accomplish by these transfusions.

Indications and Contraindications

There are five valid indications for blood transfusion:

1. To replace blood volume when it is falling or has fallen as a result of hemorrhage or burns.
2. To improve the oxygen-carrying capacity of the blood in acute and chronic anemias to alleviate symptoms of hypoxia.
3. As a method for administering substances specifically lacking, such as coagulation factors.
4. The exchange of blood in a newborn with erythroblastosis fetalis.
5. Rarely, the exchange of blood in a patient with acute infectious hepatitis in danger of death.

The diagnosis of anemia is not an indication for a blood transfusion; nor is the contemplation of a surgical operation in an anemic patient. Patients with chronic illnesses complicated by anemia such as renal disease, rheumatoid arthritis, or chronic infection, and patients with congenital anemias such as thalassemia minor are not able to withstand surgery better after transfusion. Such patients should be thoroughly studied to determine the nature of their anemia before elective surgery and should be thoroughly studied to determine their functional capacity when emergency surgery is indicated. The decision to administer blood must be made as a result of such study, not as the result of the perusal of laboratory data on the hospital chart. Patients with chronic anemia of moderate degree usually accommodate physiologically and can be counselled to adjust their living habits so as to exclude severe exertion. One of the important physiological processes in the pathogenesis of such anemias is the shortening of the red cell survival time. Thus transfusion will elevate the hemoglobin only for a short time.

Risks

There are very definite risks which accompany blood transfusions. It is estimated that six million units of blood are given each year to two million patients. Of these two million people who receive blood transfusions, 3,000 die as a direct result of the transfusion. Transfusion then would seem to be an important form of therapy but one which should not be used without weighing the possible risks against the benefits. The risks of blood transfusions fall into several categories.

1. *Bacterial contamination.* Blood is an excellent culture medium. In spite of procedures used in blood

banks for the aseptic collection of blood, one cannot guarantee that every unit of blood is sterile. Bacterially contaminated blood can give rise to chills, fever, shock, and sometimes death.

2. *Viral hepatitis.* This complication of transfusion therapy is increasing each year. It is estimated that the incidence of hepatitis from transfusions is 20,000 cases per year, of which 2,000 are fatal. In one carefully followed-up series of cases, the incidence of hepatitis was 3 percent and the overall mortality rate 0.9 percent. In patients over the age of 40, the fatality rate was 3 percent. The only way to guarantee that a transfusion will not cause hepatitis is to have the proposed recipient donate his own blood in anticipation of elective surgery.

3. *Nonspecific febrile reactions* which, while not usually fatal, result in serious discomfort to the patient. Such reactions may be the result of leukocyte incompatibility, platelet incompatibility, or allergic reactions to plasma proteins.

4. *Hemolytic transfusion reactions due to donor-recipient incompatibility.* Such reactions fortunately are rare. Most are the result of erroneous labeling of a sample sent to the blood bank for cross-matching or of inattention by personnel who give out the blood or the person who administers it so that patients are given the wrong bottle of blood.

In spite of all precautions, there are numerous instances of hemolytic transfusion reactions in which no donor-recipient incompatibility can be demonstrated in vitro prior to transfusion. In many of these cases incompatibility is demonstrated after the reaction when the titer of antibody in the recipient has been raised as a result of antigenic stimulus of the transfusion. There are a certain number of hemolytic transfusion reactions, however, which cause serious morbidity and mortality and serological testing never reveals incompatibility in vitro. In vivo survival studies in such patients sometimes show that transfused cells are quickly destroyed. Thus our knowledge of blood groups and transfusion practices is far from complete.

I cannot help quoting Crosby's comment. "Thoughtless prescription of blood transfusion is playing Russian roulette with bottles of blood instead of a revolver. While the odds are in the physician's favor that nothing will go wrong, the patient takes the risk."

(The references may be seen in the original article.)

LACERATION OF ABDOMINAL AORTA AND STUDY OF INTACT ABDOMINAL WALL AS TAMPONADE: REPORT OF SURVIVAL AND LITERATURE REVIEW

*A. J. Richards, Jr. MD, Pano A. Lamis, Jr. MD, James T. Rogers, Jr., and
Gilbert B. Bradham MD, From the Department of Surgery, Medical
College Hospital, Medical College of South Carolina, Charleston, South Carolina.
Ann Surg 164(2): 321-324, August 1966.*

Survival after laceration of the abdominal aorta is a rare and dramatic event. Cases reported to date have focused upon the technics of the surgical operation and little consideration has been given to factors influencing survival. This report concerns those features of the bleeding patient which provide a chance of successful repair. A summary of previous successful surgical repair of abdominal aortic lacerations, two case reports, and a series of experiments are presented.

Literature Review

Only 12 cases of lacerating injuries of the abdominal aorta with successful repair were found in a review of the medical literature. The first was a stab wound repaired by Wildegans in 1926, one other stab wound, one shrapnel wound and nine gunshot wounds (Table 1). It is probable that other cases of successful repair were not reported. Of 12 cases, eight have been reported since 1960. With the exception of one vitallium tube graft, all others were managed by direct suture.

Case Reports

Case 1. A 38-year-old man sustained a bullet wound of the abdomen at about 2:30 p.m., 10/24/64. The patient was then thrown from a moving automobile, subsequently found, and delivered to the Emergency Receiving Unit of the Medical College Hospital at approximately 3:32 p.m. Initially the patient was conscious, blood pressure was 62/40 and the pulse rate was 110. There was an adequate airway with good breath sounds throughout the chest. Heart sounds were distant and peripheral pulses were weak. In the abdomen there was a bullet wound of entrance anteriorly, immediately beneath the left costal margin to the left of the midline. There was no wound of exit. The abdomen was distended, tense, and there was no audible peristalsis. Rectal examination and examination of the ex-

tremities were negative. Peripheral pulses in all extremities were equal. A nasogastric tube was inserted and blood was aspirated from the stomach. A Foley catheter was inserted; there was no blood in the urine. Blood was drawn for crossmatching and appropriate intravenous fluids were given. Blood pressure rose to 110 and chest and abdominal x-rays were taken. The left leaf of the diaphragm was elevated, but the lung fields showed no damage. On abdominal films, there was a single 22 caliber bullet located posteriorly to the right transverse process of the 12th thoracic vertebra. The patient was taken to the operating room, and a large intravenous catheter was placed through a right antecubital vein into the superior vena cava. Blood was transfused and operation started at 6:00 p.m. A midline incision extended from the xyphoid process to 3 cm below the umbilicus. There were 1,000 cc of fresh blood in the peritoneal cavity. The bullet had penetrated the left lobe of the liver, the esophagus at a point just proximal to the esophagogastric junction, and the aorta in the T12-L1 level. The aortic wound was well tamponaded by the crus of the diaphragm. After dissection, fresh bleeding ensued and was controlled manually. The left hemithorax was then entered through the 7th intercostal space and the descending thoracic aorta was isolated with umbilical tape and a noncrushing arterial clamp. A second umbilical tape and arterial clamp were placed distal to the wound in the aorta to control back bleeding. With these clamps in place, the site of penetration was debrided and closed with arterial silk sutures. The distal clamp and proximal clamp were in turn removed and hemostasis was adequate. The esophageal wounds and lacerations in the liver were repaired. The chest was drained with a thoracotomy tube, and Penrose drains were placed about the liver lacerations and brought out through stab wounds in the anterior abdominal wall. The estimated blood

loss during the operative procedure of 4,500 cc had been replaced. The postoperative course was uncomplicated until the sixth postoperative day when an unexplained episode of profuse perspiration, dizziness and chest pain occurred. On the 7th day a subcutaneous infection in the thoracic and abdominal wounds was found and drained. Thereafter, the postoperative course was uneventful and the patient was discharged on the 27th postoperative day. There were excellent pulses in all extremities and there was no thrill or bruit at the time of discharge. There was no neurological deficit.

TABLE 1. *Successfully Repaired Penetrating Wounds of the Abdominal Aorta*

Authors Date	Cause of Injury	Type of Injury	Type of Repair
Wildegans 1926	Stab wound	1 cm. laceration	Suture
Dubinskiy 1944	Shrapnel wound	3 mm. laceration	Suture
Holzer 1948	Gunshot wound	5 mm. laceration	Vitallium tube graft
Holzer 1948	Gunshot wound	1 cm. laceration	Suture
Beall 1960	Stab wound	Laceration	Suture
Beall 1960	Gunshot wound	Laceration	Suture
Beall 1960	Gunshot wound	Laceration	Suture
Beall 1960	Gunshot wound	Laceration	Suture
Manlove <i>et al.</i> 1960	Gunshot wound	Laceration 6 mm. entrance, 20 mm. exit	Suture
Bradham <i>et al.</i> 1962	Gunshot wound	Laceration 1.0 X 0.5 cm. entrance and exit	Suture
Jensen 1963	Gunshot wound	5 mm. laceration	Suture
Love and Evans 1963	Gunshot wound	8 mm. laceration	Suture

Case 2. A 15-year-old boy who was in an altercation and received a gunshot wound of the abdomen from a 22 caliber rifle at 1:00 p.m. The patient arrived at the Emergency Receiving Unit of the Medical College Hospital 20 minutes later. Blood pressure was 40/20; there was a bullet wound of entrance in the epigastrium just to the right of the xyphoid process. Posteriorly the bullet could be palpated beneath the skin to the right of the first lumbar vertebra. There were decreased breath sounds in the right chest and no sounds in the abdomen. The patient was unable to move his lower extremities, and there was loss of sensation of the legs distal to the mid thigh. There was areflexia of the lower extremities and no sphincter tone on rectal examination. No blood was aspirated through a nasogastric tube. Fluids were given through a large intravenous cannula placed in an upper extremity

vein and a closed thoracotomy tube was placed in the right chest. Thereafter blood pressure rose to 100/40 and all peripheral pulses could be palpated. Paracentesis revealed fresh blood which did not clot. After blood transfusions were started, the abdomen was explored through a midline incision and a massive hemoperitoneum was immediately encountered. Fresh bleeding became evident as soon as the peritoneum was opened. Aortic bleeding was controlled by manual compression. The left chest was opened through the 7th intercostal space for control of the descending thoracic aorta. The abdominal aorta perforation, at the T12-L1 level, was controlled by tapes and arterial clamps while the penetrating wound was debrided and closed. Suture of the aorta produced an estimated 20 to 25 percent narrowing of its lumen. Upon release of the occluding clamps, there was good pulsation in the distal aorta, and good peripheral pulses could be felt. Hemostasis was adequate. The wound of the liver was debrided and closed. The left chest was drained with an underwater thoracotomy tube. Penrose drains were placed in the area of the liver lacerations. The patient was then turned to a prone position and decompression laminectomy was done for partial transection of the corda equina. On 5/11/64 suprapubic cystostomy was done to decompress a neurogenic bladder. A small sacral decubitus ulcer was surgically corrected on 6/4/64. During this procedure, there was a cardiac arrest. Cardiac function was restored by external massage and there was no additional neurologic deficit in the postoperative course. The patient was discharged on 10/3/64, having regained part of the initial neurologic deficit, with good arterial supply to extremities, and all wounds healed.

Experimental Studies

Ten mongrel dogs were lightly anesthetized with sodium pentobarbital. Electrocardiographic leads were attached to the extremities. Both femoral arteries were cannulated proximally so as to place the tips of vinyl catheters at a standardized location in the mid-abdominal aorta. One catheter was used to monitor aortic blood pressure; the other was used for bleeding purposes. The ten dogs were randomly divided into two equal groups. In Group I, blood was allowed to flow freely out of the vinyl catheter tip held at the vertical level of the heart. This blood flowed into a graduated receptacle and was propelled only by the difference between the intraluminal pressure of the abdominal aorta and the ambient atmospheric pressure. In Group II, the tip of the vinyl catheter was implanted into the peritoneal

cavity through a small stab wound in the right lower quadrant of the abdomen. The abdominal wall was sutured tightly about this end of the catheter (Fig. 1, not shown). Blood from both groups of dogs was allowed to flow freely until death occurred as evidenced by an asymptotic EKG wave.

Results. Mean survival time of the five animals in Group I was 17.6 minutes (S.D. \pm 4 minutes). The mean survival time of the five animals in Group II was 96.2 minutes (S.D. \pm 44 minutes).

The standard error of the difference between the means of the two groups was 19.92, giving a confidence limit above the 99 percent level that this difference was not by chance.

Discussion

The factors which provide for successful surgical repair of aortic lacerations can be grouped under these headings.

1. *Transit Time from the Scene of the Accident to the Emergency Room.* This factor has been emphasized previously and needs no elaboration. Of 384 postmortem cases of penetrating wounds of the heart and aorta, only 12.2 percent survived for one hour.

2. *Prompt Availability of Blood.* The adequately administered emergency unit should have proximity to a well organized blood bank to dispense type specific blood. However, an immediate source of O-type blood is mandatory to treat successfully patients with serious large blood vessel injuries.

3. *Transit Time to the Operating Room.* Once the patient has received emergency care, the time elapsing prior to operation becomes crucial. There should be no delay for procedures other than mechanical control of hemorrhage from the aorta. X-rays may be required during transit from the emergency room to the operating room.

4. *Tamponade.* Tamponade may account for survival from a laceration of the aorta and more than any other single factor. "The surgeon should be aware that there may be circulatory collapse when the blood under tension in the peritoneal cavity is released as the abdomen is opened." In Case 1 here reported, tamponade was effected by the vertebral column and its ligaments posteriorly, and by the musculature of the diaphragm anteriorly. Once

this tamponading effect was disrupted, blood loss was greatly accelerated until clamp control could be initiated. This tamponading acts as an *internal hemostat*.

In the experiments, an important clinical fact was demonstrated. Animals survive longer if they bleed into the peritoneal cavities rather than exteriorly, due to the tamponading effect of the intact abdominal wall. Also important is the fact that the peritoneum can reabsorb plasma and red cells. If this absorption occurred rapidly, the effect would be autotransfusion during hemorrhage. This factor is difficult to assess.

On this basis, in a patient with large vessel injury in the abdomen, an incision may accelerate deterioration of the patient. A large amount of blood should be available with apparatus for warming the blood. Warm blood transfusions in large amounts are accompanied by fewer cardiac arrests than are cold blood transfusions. A pump for forceful transfusion should be available.

5. *Diagnosis.* While diagnosis of aortic injury is difficult, knowledge of the conditions of the trauma is important along with thorough physical examination, evaluation of the preoperative status of the patient and estimate of blood loss.

Summary

Survival following lacerations of the abdominal aorta is infrequent. Only 12 cases of successful repair of aortic lacerations have been reported.

Two additional cases of survival following gunshot wounds of the abdominal aorta are reported. These cases direct attention to various factors contributing to successful repair of the aorta: 1) transit time from the scene of the accident to the emergency room, 2) prompt availability of blood, 3) transit time to the operating room, 4) tamponade and 5) diagnosis.

Experiments designed to simulate lacerations of the aorta are presented. These experiments demonstrate the effect of the intact abdominal wall as a tamponading element.

(The references and omitted figure may be seen in the original article.)

See "The Use of the G Suit in Control of Intra-Abdominal Bleeding", W. J. Gardner MD and J. Storer MD, Surg Gynec Obstet 123(4) : 792-798, October 1966.

MEDICAL ABSTRACTS

STUDIES IN HUMAN PHYSIOLOGY INCIDENT TO EXTENDED SURGERY FOR CANCER

Alexander Brunschwig MD, (From the Memorial Hospital for Cancer and Allied Diseases and The James Ewing Hospital of the City of New York. This constituted The James Ewing Lecture at the 19th Annual scientific session of the James Ewing Society, 21 April 1966.) Cancer 19: 1479-1485, November 1966.

Dr. Brunschwig, in this paper, has reviewed physiological observations which he has recorded in more than 20 years operative experience concerned mainly with cancer. In the introduction, he reminds us that some operations, performed as a result of the expanded surgical attack upon cancer, may result in profound anatomo-physiological alterations that lend themselves to studies not heretofore possible in man. Subject headings are: neurogenic factors in surgical shock; nutrition by intravenous alimentation; gradual versus sudden assumption of physical load and the stimulus to hyperplasia and hypertrophy; gastric secretory depressant in cancerous stomachs; possible existence of a factor in gastric mucosa that contributes to general nutrition; radioactive isotopes in human physiological studies of gastric secretion; physiology of the pancreas; total pancreatectomy in man; ligation of the pancreatic ducts; successful animal experiments suggesting specific cytonecrotic effect that failed on clinical application; ligature transection of the portal vein; duration of acute occlusion of the ureter and recovery of renal function; deperitonealization and reperitonealization; maximal body temperatures compatible with human life; continual survival following multi-visceral excisions.

He urges continuation of such studies when the opportunity presents.

THE DIFFERENTIATION BETWEEN ESOPHAGEAL AND CARDIAC PAIN

*J. R. Bennett MD and M. Atkinson MD,
Lancet II: 1123-1127, Nov. 19, 1966.*

The differentiation is not easy. The authors made a prospective survey of all patients admitted to a

general medical unit because of precordial pain; 23 percent had only alimentary-tract disease, mostly esophagitis. The clinical features of esophageal pain may resemble those of cardiac pain closely and there may be identical radiation and precipitation by exercise or emotion. Age, site of pain, type of pain, precipitating factors (exercise, posture, emotion, meals), relieving factors (rest, antacids, glyceryl trinitrate), were evaluated in all patients as were associated symptoms—(dyspnea, faintness, oral reflux, heartburn, vomiting, sweating), and physical findings. Electrocardiograms were abnormal in all patients with myocardial infarcts and in all but two with angina pectoris; in esophagitis, only four of 39 records were abnormal, two showing ischemia and two nonspecific abnormalities. Barium meal studies in 37 percent of those with an eventual diagnosis of esophagitis were normal; 46 percent had an esophageal hiatus hernia; three peptic ulcers were found, and the remaining patients in the group showed gastroesophageal reflux, esophageal spasm, or “tertiary contractions”. Only 13 patients with ischemic heart disease had a barium meal, five were normal and there was a hiatal hernia in seven.

The investigation has revealed the importance of esophageal disease as a cause of precordial pain.

PULMONARY MICROEMBOLISM—A CAUSE OF MORBIDITY AND DEATH AFTER MAJOR VASCULAR SURGERY

F. W. Blaisdell MD, R. C. Lim, Jr. MD, J. R. Amberg MD, S. H. Choy MD, A. D. Hall MD, and A. N. Thomas MD, (From the Departments of Surgery, Radiology, and Pathology, Veterans Administration Hospital, and University of California School of Medicine, San Francisco.) Arch Surg 93: 776-786, Nov 1966.

In reviewing 400 major vascular reconstructive procedures at San Francisco Veterans Administration Hospital in the past six years, it was found that most of the serious morbidity and mortality occurred in emergency cases. There were 25 deaths in 106 emergency operations for aneurysm and occlusive disease and only six deaths in 269 elective operations of the same type. The most significant finding was

that 18 out of the 31 deaths were related to cardiopulmonary complications. Excluding eight immediate deaths from exsanguination (ruptured aneurysms) three fourths of the postoperative deaths from arterial reconstruction for disease of the infrarenal aorta or its branches were due to problems with the heart and lungs but not congestive failure, myocardial infarction, or pneumonia although these were the final definite diagnoses. A similar clinical picture was noted in half of the survivors of emergency surgery for occlusive disease. A review of the clinical course, laboratory, x-ray film, and pathologic findings in this group indicated that the common etiologic factor in most was pulmonary microembolism.

Fourteen of the patients developed the complications which the authors call the syndrome of pulmonary microembolism. The characteristics are described as: (1) local shock, (2) bleeding problems, (3) serious respiratory disturbances with cyanosis and arterial desaturation, and (4) manifestations of low cardiac output. Pulmonary changes were noted in x-ray films, but there were no characteristic lesions. At autopsy, fibrin emboli were found in pulmonary arteries 50 to 200 μ in diameter in all patients who died in the first three days and none in the three who died five days or more following surgery. Prevention, they say, involves the avoidance of prolonged regional shock, vigorous treatment of hypotension, and immediate anticoagulation with heparin. Treatment includes tracheostomy, ventilation with 100 percent oxygen, heparinization, and careful monitoring of arterial oxygen and pH.

The authors think that pulmonary microembolism may be a frequent cause of morbidity and death following emergency operation for arterial occlusive disease or ruptured aneurysm and that awareness of the cause, prevention and treatment of this syndrome should lower morbidity and mortality after major vascular surgery.

CASTRATION AS PART OF THE TREATMENT FOR FEMALE BREAST CANCER— A STATISTICAL EVALUATION OF CLINICAL RESULTS

Roan Nissen-Meyer, (From the General Department [Director: Professor E. Poppe], The Norwegian Radium Hospital, Oslo, Norway.) *Acta Radiologica Supplementum* 249, Stockholm, 1965.

This supplement, some 133 pages including the appendix and tables, records an investigation with

the following aims: (1) to test statistically the significance of the effect of primary ovarian radiation for operable breast cancer, (2) to compare statistically the effects of primary ovarian irradiation and primary bilateral oophorectomy, and (3) to study in more detail the effect of primary castration in different age groups and different prognostic groups of operable breast cancer patients.

In Chapter I, the author has reviewed the literature on the subject including prophylactic versus therapeutic castration and surgical versus radiological castration and to some degree, on chemotherapy as adjunct to primary treatment for breast cancer. In Chapter II, he discusses problems and plan of the present investigation, in Chapter III, material and methods, in Chapter IV crude survival rates in relation to stage, in Chapter V, he presents statistical tests of the effect of primary ovarian irradiation and oophorectomy upon rates free of disease and crude survival rates, and in Chapter VI, rates free of disease and crude survival rates in relation to prognostic factors and primary castration. Chapter VII includes a general discussion and his conclusions. Chapter VIII is a summary and this is followed by references and appendix-tables.

The following practical conclusions are listed:

"The advantages of primary castration have to be balanced against the disadvantages of the treatment, and against the chance that the patient has already been cured by the conventional treatment.

In postmenopausal patients, the disadvantages so far observed seem negligible. In the Norwegian Radium Hospital we therefore now consequently give primary ovarian irradiation to postmenopausal patients up to the age of 70 years.

If the patient is premenopausal, but 45 years old or more, we also recommend primary ovarian irradiation to patients with stage I as well as stage II cancers.

If the patient is younger than 45 years, we normally recommend ovarian irradiation to patients with stage II cancers. To young patients with a stage I cancer we are more reserved, especially if the malignancy grade is low and the tumour was laterally located. If the patient is reluctant, we would not try to persuade her. On the other hand, many patients have no objection to ovarian irradiation, especially when a new pregnancy is not wanted.

In this hospital ovarian irradiation is now used for castration instead of oophorectomy, except in the cases where a quick response is wanted."

WHITE BLOOD CELL ANTIBODIES— OCCURRENCE IN PATIENTS UNDERGOING OPEN HEART SURGERY

B. G. Hattler, Jr. MD, W. G. Young, Jr. MD, D. B. Amos MD, P. Hutchin MD, and M. MacQueen, (From the Division of Thoracic Surgery, Department of Surgery, and the Division of Immunology, Department of Microbiology, Duke University Medical Center, Durham, N. C.) *Arch Surg* 93: 741-746, November, 1966.

In the introduction to this article, the authors note that leukocytes are known to carry transplantation antibodies in animals and in man and they raise the question: "Should transfusions of blood from one individual to another, therefore, be considered as a homograft and to what extent do blood transfusions sensitize for a subsequent graft." Since leukocyte antibodies have been demonstrated following massive transfusions of fresh blood containing living leukocytes (leukocytes are not well preserved in stored blood), serum from 24 patients undergoing heart surgery was examined during the preoperative and postoperative period following cardiopulmonary bypass to investigate further the incidence of leukocyte antibodies in patients undergoing open heart surgery, to correlate, if possible, the appearance of such antibodies with certain postoperative syndromes which may have an immunologic basis, and to determine to what extent a transfusion of fresh blood will sensitize an individual to a subsequent skin graft.

Potent lymphocytotoxic antibodies developed in seven patients; six of nine adult female patients examined developed antibodies but only one of the 12 males in the study demonstrated a similar response. In other experiments, eight normal subjects demonstrated accelerated skin graft rejection two weeks following exchange of one unit of fresh blood.

The authors warn that the use of fresh whole blood, because of the known sharing of antigens between leukocytes and many tissues, may be inadvisable in patients who may later be considered as homograft recipients.

STRANGULATION AND OBSTRUCTION IN DIAPHRAGMATIC HERNIA DUE TO DIRECT TRAUMA

R. E. Sullivan MD, (From the Waterbury Hospital, Waterbury, Conn.) *J Thorac Cardiovasc Surg* 52: 725-734, Nov 1966.

Diaphragmatic hernia due to indirect violence such as falls and automobile injuries has been reported frequently but only 53 cases of obstruction and strangulation in diaphragmatic hernia due to penetrating injury had been reported in the English literature before this review according to Dr. Sullivan. He has reviewed all the cases reported and added two more. The majority of these resulted from stab or gun shot wounds or followed hiatal hernia repair.

The colon herniated most frequently followed closely by the stomach. All occurred on the left side (liver occludes small tears on the right). A trans-thoracic route was used in the repair of 30 cases, laparotomy was done in 15 and a combined thoraco-abdominal approach was used in four. The overall mortality rate was 7.4 percent and operative mortality 4 percent. The interval between injury and strangulation varied from a few hours to 30 years but most often was two to three years. The common occurrence of strangulation and obstruction has been attributed to the small size of the tear in the diaphragm, plugged initially by omentum, but because of the negative intrathoracic pressure and high intra-abdominal pressures, viscera herniate through the rent and become strangulated.

Additional points stressed in the author's summary are: consider the diagnosis of traumatic diaphragmatic hernia in any patient with a history of or visible signs of previous trauma associated with signs and symptoms of gastrointestinal obstruction; the occurrence of dyspnea and mediastinal shift suggests massive herniation of abdominal viscera or the accumulation of large amounts of fluid in the pleural cavity; the chest x-ray may be extremely misleading; mortality rates following penetration of the diaphragm have been reduced considerably with increasing awareness of the diagnosis of strangulation and obstruction in diaphragmatic hernia and improved surgical techniques.

DENTAL SECTION

EXPANDED DENTAL PROSTHETIC CAPABILITY PROGRAM

It has become increasingly evident that a system of support must be provided to smaller dental facilities with no prosthetic capability. The establishment of a prosthetic facility at each activity, with its attendant cost in money and trained laboratory personnel, appeared not only impractical but wasteful. In order to obviate the time lost in patient travel it was deemed necessary to provide at each station a limited prosthetic capability where impressions could be made, try-ins accomplished and the completed denture delivered. To support this concept, it became evident that a central or area laboratory must be used for the fabrication of the dentures and concentrate herein the equipment and personnel talent needed.

A feasibility study was inaugurated in September 1964 in the Washington Naval District to test the validity of this concept and to determine the professional acceptance of the end product. In 1965, upon completion of the feasibility project, it was determined the concept was valid and the end product professionally acceptable. A bonus effect achieved during the pilot study was noted in that the area laboratory could render support on an interim basis to established dental prosthetic laboratories. Once the area laboratory is geared to production, it can absorb fabrication of cases from temporarily overloaded established prosthetic laboratories and help to reduce a potential back-log.

The Limited Dental Prosthetic Laboratory: The limited capability dental prosthetic laboratory (LDPL) provides the armamentarium to take impressions, pour models and casts, survey the case, select molds and shades, repair dentures, invest inlays and crowns, and insert finished dentures.

No duplicating, burn-out, or casting equipment is provided and no prosthetic technicians are furnished.

The LDPL is supported by a dental prosthetic laboratory (DPL) or an area dental prosthetic laboratory (ADPL) upon which it is dependent for

wax-up, casting, and finishing; the actual fabrication of the prosthetic appliance. Transmission of casts, frame-works, try-ins, etc., is accomplished by guard mail or U.S. Mail, whichever is more expeditious.

Approximately 50-60 square feet are needed in addition to normal size operator (115 sq ft). The limited dental prosthetic capability can be accommodated in an over-sized operator, or temporarily, in a normal sized operator.

The concept of the LDPL is to provide a limited prosthodontic capability to those smaller and more isolated activities that do not warrant the investment of equipment or personnel, due to their limited base-loading of personnel.

The Dental Prosthetic Laboratory: The dental prosthetic laboratory, or DPL, is the prosthetic laboratory we have had so long in the Navy and provides a full prosthetic capability, with the necessary equipment and personnel assigned.

The Area Dental Prosthetic Laboratory: The area dental prosthetic laboratory (ADPL) provides a complete prosthetic laboratory capability and contains specialized equipment and personnel. It provides laboratory support to several activities and is designed for production line fabrication of prosthetic appliances.

The ADPL came into being to support the LDPL concept in order to absorb the additional denture fabrication work-load. It provides a concentration of expensive specialized equipment and highly trained prosthetic technicians. ADPL's support LDPL's, and some DPL's continuously, and some DPL's on occasions.

Conclusion: The LDPL and ADPL concept provides a greater prosthetic capability Navy-wide at a considerable saving in patient man hours formerly lost in transit and a savings in prosthetic technician man hours. It is based on the realization that it is more efficient to transport the material from site to site than personnel from site to site.

PERSONNEL AND PROFESSIONAL NOTES

IN MEMORIAM

RADM Daniel W. Ryan DC USN (Retired) died Sunday, November 13, 1966 at U.S. Naval Hospital, Bethesda, Maryland, after a short illness. He was 66 years of age.

Doctor Ryan was born in Iowa and received his DDS degree from the University of Denver in 1923. He entered the Dental Corps of the U.S. Navy from Manchester, Iowa in 1925 and served at sea in the USS LEXINGTON, USS RIGEL, the Hospital Ship USS RELIEF and overseas in the Philippines and China. He also served in numerous and important shore activities in the United States. He was appointed to the grade of Rear Admiral in March 1950 and served as Chief of the Dental Division, Bureau of Medicine and Surgery from 1952 to 1955.

Among his numerous honors, Admiral Ryan was a Fellow of the American College of Dentists, and a Fellow of the International College of Dentists.

Admiral Ryan is survived by his wife, Marjorie and a daughter, Mrs. Glen M. Eben. Services were conducted at the Greenwood Mortuary, San Diego and interment on Friday, 18 November at the Greenwood Memorial Cemetery.

CERTIFICATE OF HONOR FOR EXHIBIT

The American Dental Association awarded the Dental Corps of the U.S. Army, Navy, and Air Force, the Certificate of Honor for the joint scientific exhibit at the annual session of the ADA, November 13-17, 1966 in Dallas, Texas.

The exhibit entitled, "Advanced Concepts of Operative Dentistry," is the second joint exhibit produced through the cooperative effort of the Joint Armed Forces Dental Exhibit Committee with chairmanship rotating each year. The U.S. Air Force held the chairmanship of the committee re-

sponsible for this year's exhibit. Constructed at the Armed Forces Institute of Pathology, the exhibit was based upon the U.S. Naval Dental Corps' motion picture, "Advanced Concepts of Operative Dentistry." The joint effort between the three services was promulgated as an economy measure and to establish a closer working relationship between the three services. Its goal was to contribute to the continuing education of the professional man.

NAVY MOTION PICTURE AWARD

The U.S. Naval Dental Corps received an Honor Award for the production of a new motion picture, "Advanced Concepts of Operative Dentistry." The citation was bestowed by the Association of Military Surgeons of the United States during its Annual Meeting held at the Washington Hilton Hotel, Washington, D.C., November 7-9, 1966. The motion picture stresses a comprehensive plan of treatment wherein operative dentistry is considered a part of the total health program.

NOTICE TO ALL USERS OF POWERAIR STRAIGHT HANDPIECES

The Ritter Equipment Company, P.O. Box 848, Rochester, New York 14603, has made available to the Armed Services, a hexagonal-nose coupler for use with the Powerair straight handpiece. The hex-nose coupler permits the use of existing standard stock hexagonal-sleeve, contra-angles with the Powerair straight handpiece for slow-speed procedures. The coupler and coupler chuck key can be procured, open purchase, direct from the Ritter Equipment Company:

<i>Item</i>	<i>Price</i>
#8 Hex-nose coupler (#195601)	\$19.50
Coupler chuck key (#136302)	2.28

12TH DENTAL COMPANY MAKES FIELD EXERCISE "PAY OFF" FOR CHILDREN

The 12th Force Dental Company, 2d Marine Aircraft Wing, Cherry Point, N.C., CAPT L. Young DC USN commanding, executed an annual field training exercise which accomplished two additional commendable values. To accommodate these special purposes, the field exercise was established adjacent to the station's main baseball diamond, vice the usual remote site. In three platoons, 11 dental officers and 17 dental technicians established a field complex of three GP and two CP tents for the period 12-19 August, 1966, for the primary purposes of training newly assigned personnel in mount-out procedures and testing field equipment and procedures.

After the Oral Surgery, the Prosthodontic and the Clinic Platoon had completed their field exercise, all hands turned to for four days and provided three-agent stannous fluoride preventive dentistry treatment and oral hygiene instruction to 1430 school aged dependent children of military personnel attached to units at Marine Corps Air Station, Cherry Point. This accomplishment is especially impressive in that the conventional rubber cup prophylaxis was included. During this exercise, the 12th Dental Company also celebrated the 54th anniversary of the Naval Dental Corps. To the officers and men of the 12th Dental Company, a hearty "Well Done."

NURSE CORPS SECTION

INTENSIVE CORONARY CARE WORKSHOP

*LCDR Ruth Pampush NC USN, Educational Coordinator, Nursing Service,
Oak Knoll Naval Hospital.*

An intensive coronary care workshop for nursing service has been established at this hospital under the supervision of CAPT Henry A. Sparks, Chief of Medicine for the purpose of instructing staff in the performance of duties in the intensive coronary care unit which is now functioning at Oak Knoll.

The purposes of the coronary care unit are to improve the medical and nursing care of the patients with recent or suspected myocardial infarction and to decrease the number of resultant deaths, to bring medical and nursing practice to a level where it utilizes the knowledge gained from research, and to help fulfill our obligation to the American people who have donated millions of dollars to hear research and who have been taxed for millions more.

The concept of the coronary care unit is therefore a natural product of current needs and current capabilities. The unit is a separate area within the hospital specifically equipped and staffed to meet the total anticipated needs of the patient with myocardial infarction. Constant, intensive surveillance is provided, and emergency treatment can be instituted

without delay. Although the facility is equipped to monitor electrocardiograms and other vital signs as indicated, no degree of electronic excellence can replace the nurse and physician.¹

Because she is the one who is with the patient for twenty-four hours a day, the nurse specialist is the most vital element in the whole concept of intensive coronary care. She offers excellence in intensive nursing procedures through her familiarity with the particular physical and emotional needs of the coronary patient and through her training in the recognition and emergency management of complications.²

The present workshop together with a continuous teaching program for constant retraining of intensive coronary care personnel has been developed to produce the nurse specialists who will work in the unit.

Objectives of the workshop are: to gain knowledge and understanding of cardiac function, cardiac disease, diagnostic measures used in detecting cardiac

1. "Training Technics for the Coronary Care Unit," Second Bethesda Conference of American College of Cardiology, Dec. 11, 12, 1963, Washington, D. C., American Journal of Cardiology, May 1966, p. 739.

2. Ibid.

disease, cardiac drugs, electronic machines, comprehensive nursing of cardiac disease patients, the nurse's role as teacher; and the development of special skills used in the intensive coronary care unit. The special skills include observation skills, ECG interpretation—identification of patient problems as evidenced in the ECG tracing, proper use of technical nursing skills, proper use of communication skills, proper use of medical-scientific vocabulary, and patient teaching.

Course content includes the following; purposes, aims & objectives, statistics, anatomy of cardiac circulation, pathology of myocardial infarction, enzymes involved in myocardial infarction and its detection, clinical picture of the patient with a myocardial infarction, coronary precautions, monitor units—use, function, hazards, ECG apparatus and function, practice using ECG, monitors, principles of ECG pattern recognition, normal readings, arrhythmias, cardiac drugs, cardiac resuscitation,

demonstration and practice of defibrillation in the dog lab, airways, respirators, tracheal toilet, metabolic factors in cardiac arrest, pacemakers, shock, and the unit drill of precise roles in cardiac arrest.

The workshop was developed by CDR W. S. Myers MC USN, Assistant Chief of Medicine and Cardiologist, LT Gerald A. Wolff MC USN, Cardiologist, and LCDR Ruth G. Pampush NC USN, Nursing Educational Coordinator at Oak Knoll, under the direction of LT Wolff who had worked with Dr. Bernard Lown in establishing the Samuel A. Levine Cardiac Center at Peter Bent Brigham Hospital in Boston. Many instructors from other services contributed their time and the cardiologists, in particular, spent many hours assisting with ECG pattern recognition drill.

The enthusiasm of the students and commitment of the instructors resulted in a highly successful and rewarding workshop. It will be repeated 14-19 November to augment the present ICCU staff.



DEPARTMENT OF DEFENSE NURSING ADVISORY COMMITTEE MEETS

The first meeting of the Department of Defense Nursing Advisory Committee convened on 14 October 1966 at the Pentagon. Members present included:

Shirley C. Fisk MD, Deputy Assistant Secretary of Defense (Health and Medical).

COL Mildred I. Clark NC USA, Chief, Army Nurse Corps.

CAPT Veronica M. Bulshefski NC USN, Director, Navy Nurse Corps.

COL Ethel R. Kovach NC USAF, Chief, Air Force Nurse Corps.

CDR Alene Duerk NC USN, Special Assistant for Nursing Affairs, OSAD(M), (Health and Medical).

Mrs. Judith Whitaker, Executive Director, American Nurses' Association.

Mrs. Lulu Hassonplug, Dean, School of Nursing, University of California, Los Angeles.

Mrs. Joyce T. Via, Executive Secretary, OASD (M), (Health and Medical).

A general discussion was held concerning nursing problems pertinent to military and civilian nursing. The directors of the military nurse corps presented a synoptic profile of their respective corps. The group arrived at an agreement concerning the following definitions of the various levels of nursing practiced by military and civilian nurses in military medical activities and the minimum requirements for each level:

Professional Nurse—Graduation from an approved nursing education program providing a minimum of three academic years, exclusive of vacations.

Current licensure in nursing in a state of the United States, District of Columbia, or Commonwealth of Puerto Rico.

Technical Nurse—Graduation from a basic program in nursing from a school of nursing which offers not less than a two academic year curriculum exclusive

of vacations and which awards either an Associate of Arts degree or a diploma in nursing.

Current licensure as a practitioner of nursing in a state of the United States, District of Columbia, or the Commonwealth of Puerto Rico.

Vocational Nurse—Completion of a formal program of vocational nursing of not less than one calendar year duration and currently licensed to practice vocational nursing in the same states as above.

NEWLY SELECTED NURSE CORPS COMMANDERS MEET

A short course, "Nursing Service Administration", was conducted at the U.S. Naval Medical School, National Naval Medical Center, Bethesda, Maryland from 17 through 21 October 1966. The program was conducted for twenty-seven recently selected Commanders of the U.S. Navy Nurse Corps and included speakers who presented papers on administration, supervision, education, research guidance, and counseling. The theme of the entire program was directed toward assisting the new Commanders to assume their future roles as leaders in Nursing Service.

PREVENTIVE MEDICINE SECTION

INFLUENZA: PREPARATION FOR THE 1966-67 SEASON

WHO International Influenza Center for the Americas operated by the Public Health Service, Communicable Disease Center, Virology Section, Atlanta, Georgia 30333.

Despite the influenza immunization program carried out in accordance with paragraphs 12b(5) of BUMED Instructions 6230.1D, 1965, sporadic outbreaks of influenza may be expected to occur this coming winter in the naval service.

The following provides up-to-date information on influenza detection:

Type A2 and type B influenza viruses isolated during 1965 from many geographic areas showed continued antigenic variation from the A2/Japan/305/57 and B/Maryland/1/59 strain. Because of the length of time which has elapsed since the emergence of the Asian influenza virus, another major antigenic shift might be expected at any time in the

future and the coming season is an important one for laboratories collaborating in the WHO Influenza Program.

The success of this program, particularly in the detection of variant viruses, is dependent upon the rapid reporting of outbreaks and submission of isolated viruses to the International Center. It is requested that all laboratories notify the Virology Section, Communicable Disease Center, Public Health Service, Atlanta, Georgia 30333, with copy to BUMED (Code 72) when outbreaks of respiratory disease occur in their areas, even though the etiology may not have been established. Isolated influenza viruses should be submitted promptly for antigenic

comparison with viruses isolated in other geographic areas and in earlier years in order that maximum time will be available for consideration of new vaccine strains and for production of new diagnostic reagents if needed.

Kits of diagnostic reagents together with instruction for their use, will be distributed within the next few weeks to laboratories throughout the world participating in the WHO Influenza Program. This year, hyperimmune type A2 and type B chicken antisera will be included in the kit. It should be noted that hyperimmune antisera tend to minimize the differences among strains and therefore representative new isolates should be promptly submitted for more complete antigenic analysis. Because of the broad reactivity of these preparations it should be possible to identify any prevalent influenza virus by hemagglutination inhibition test unless there is a major antigenic change. If isolates are not identifiable by this test, animal antisera are also included which have titers against the group soluble antigen, and isolates may be identified as group A or B by the complement fixation test. Failure to identify a new isolate using the chicken antisera provided, and subsequent identification of the virus as either group A or B by complement fixation test, probably indicates major antigenic change. This should be a cause for immediate concern and the Communicable Disease Center, Atlanta should be notified promptly.

In recent years, type B influenza viruses have been found to be extremely sensitive to nonspecific inhibitors in sera used in the hemagglutination inhibition test. Heat, trypsin and potassium periodate treatment, as well as treatment with carbon dioxide have not been completely effective for removal of these inhibitors. On the other hand, receptor destroying enzyme (RDE) has been quite satisfactory and this reagent, with details for its use, will be included in the kits. RDE is equally satisfactory for removal of inhibitors to which type A viruses are sensitive.

Requests for additional reagents, assistance in characterizing outbreaks, or assistance in the solution of laboratory problems should be directed to the International Influenza Center for the Americas, Communicable Disease Center, Atlanta, Georgia. In order that all laboratories may be kept informed of the most recent developments throughout the world, all pertinent epidemiological information, as well as information on technical laboratory problems, will be published in the CDC Influenza Res-

piratory Disease Surveillance Reports, a copy of which is sent to your laboratory.

HUMAN RABIES DEATH—SOUTH DAKOTA

CDC Morb & Mort Wkly Rpt 15: 38, Sept 24, 1966.

A 10-year-old boy from Bryant, South Dakota, died of rabies on 5 September 1966. On 3 August the boy had been sleeping in his parents' backyard in a sleeping bag. He was awakened when a striped skunk bit him on the right thigh after apparently crawling into the sleeping bag with the boy. While attempting to get away from the skunk, the boy received additional bites on the wrist, the fingers of both hands, and behind the right ear.

The skunk escaped, but what is believed to be the same animal was shot several hours later by the boy's father. This skunk was confirmed as rabid by Seller's stain and direct fluorescent microscopy procedures at the South Dakota State Veterinary Diagnostic Laboratory, Brookings, South Dakota.

A local physician cleansed the child's bite wounds with phisohex and water and then painted them with tincture of merthiolate. A booster dose of tetanus toxoid was given at that time.

Eleven ml of antirabies serum was given within 18 hours of the exposure. Approximately 1/2 of the volume was infiltrated around the bite wounds and 1/2 injected intramuscularly. The child was started on duck embryo origin rabies vaccine the same day and thereafter received a 1 ml dose daily for the following 21 days. During this course of treatment, he received Benadryl, 50 mg q.i.d.

Twenty-four days after the exposure, the boy developed a severe headache. There were no prior symptoms except for a "funny feeling" in the fingers of the right hand before onset of headache. He was hospitalized at DeSmet, South Dakota, the following day when he developed a fever of 104°F. About 48 hours after the onset of headache he became irrational. There was a short period of hyperexcitability, laryngeal spasm and increased salivation, followed by coma.

The boy was transferred to Sioux Valley Hospital, Sioux Falls, South Dakota, on 1 September 1966. At this time he responded only to deep pain. Deep tendon reflexes were diminished, more so in the upper than lower extremities. He remained comatose until death on 5 September 1966.

Therapy included ACTH, instituted early in the course of illness because of the possibility that

symptoms might be a vaccine reaction. Subsequent treatment included tracheostomy, steroids for hypotension and urea to reduce cerebral edema.

Tissues were submitted to the State Health and CDC Laboratories for microscopic examination and virus isolation. Impression smears from the brain, lungs, and salivary glands were negative on direct fluorescent microscopic examination. A positive virus isolation was made in mice and the brains from the first mouse passage, were positive by direct fluorescent microscopy for rabies. Preliminary immunologic tests on blood serum drawn terminally, using the indirect fluorescent antibody technique, were positive.

Editorial Note: This clinical failure illustrates the limitation of present rabies prophylactic procedures. In spite of nearly ideal management including thorough cleansing of the wounds, infiltration with anti-rabies serum and a full course of vaccine, the patient developed rabies in less than 30 days from time of the bites. Skunks are known to excrete higher titers of virus in their saliva than other rabid animals. Bites involving the fingers and face, anatomical areas heavily supplied with nerve endings, are known to carry a greater hazard of disease.

THREE OUTBREAKS OF FOODBORNE DISEASE WITH DUAL ETIOLOGY

Donald R. Peterson MD MPH, Herbert W. Anderson BS and Roger Detels MD, Public Health Reports, U. S. Department of Health, Education, and Welfare 81(10): 903-904, October 1966.

In December 1965, 3 consecutive outbreaks of foodborne disease were traced to 3 banquets held at

a single restaurant in Seattle, Washington. The investigation of these outbreaks was conducted in two phases. The outbreak after the last banquet was investigated first; then 3 weeks later the outbreaks after all 3 banquets were investigated.

Information was obtainable, by telephone interview, from 345 of the 375 persons attending the banquets. The attack rate after the 3 banquets combined was approximately 40%; 140 persons were ill. Illness frequency increased after each successive banquet. The attack rate after the first banquet was 23% and after the third banquet, 69%. Two persons were hospitalized with severe illness. An estimated 506 person-days of illness, or between 3 and 4 days for each person, resulted from the epidemic.

Both *Clostridium perfringens* and *Salmonella typhimurium* were implicated as agents of infection in the 3 outbreaks. The vehicle of infection was inadequately cooked and stored turkey meat. Several unique epidemiologic features ascribed to both dual etiology and temporal sequence of exposure were noted. The mean incubation period was intermediate between that typical of infection with either agent alone. The distribution pattern for duration of illness was dissimilar to that usual with infection from either agent separately. Symptoms of abdominal pain, diarrhea, and headache were particularly persistent.

All 5 isolates of *C. perfringens* were heat-resistant, type A organisms. Eleven of 12 salmonellae isolates were *S. typhimurium*, phage type 2b. Double infection was confirmed bacteriologically in 3 patients. *C. perfringens* were isolated 6 days after the onset of illness in one instance and 7 days after in another.

DIPHTHERIA

USDHEW PHS CDC Diphtheria Surveillance Rpt No. 7, June 1966.

While in 1964 there were 293 cases of diphtheria with 34 deaths in the United States, this was a decline of 6.7% from 1963, when 314 cases and 45 deaths occurred. Since 1933, the first year of complete death-registration by State, the morbidity and mortality of diphtheria fell dramatically from

1933 to 1950, leveling off in recent years. The case fatality ratio, however, has not been appreciably lowered, and in fact it is somewhat greater in 1961, 1963 and 1964 than in any of preceding years as seen in Table 1.

Table 1
Diphtheria Morbidity and Mortality in the United States for Selected Years

YEAR	CASES	DEATHS	Rates per 100,000 Population		Case Fatality Ratio
			Case	Death	
1933	50,462	4,937	40.1	3.9	9.8
1940	15,536	1,457	11.8	1.1	9.4
1950	5,931	410	3.9	0.3	6.9
1960	918	69	.51	.04	7.5
1961	617	68	.34	.04	11.0
1962	444	41	.24	.02	9.2
1963	314	45	.17	.02	14.3
1964	293	34	.15	.02	11.6

PROBLEMS WITH WATER

*Commander Service Force, U. S. Pacific Fleet
Information Bulletin, August 1966.*

Practically all water that appears in public or private supplies has been exposed to pollution either while running over the ground surface, flowing in streams, or percolating through soil. All water received by Naval installations is adequately treated so that it is safe for human consumption. However, the purity of water must never be taken for granted by those responsible for its quality. For this reason, bacteriological studies of water must be performed on ALL SHIPS AND STATIONS OF THE NAVAL ESTABLISHMENT AT REGULAR INTERVALS. It is the responsibility of the Medical Department Representative to ascertain that the potable water is safe for human consumption.

Since it is known that a large portion of the population are carriers of salmonella, shigella, typhoid or amoeba in their intestinal tract, fecal contamination of water could easily be the cause of an outbreak of disease. For this reason, bacteriological examination of water is done to determine only fecal contamination, following the thought that any evidence of fecal contamination indicates the possibility of enteric pathogens being present. The intestinal tract of man is loaded with a group of organisms known as coliforms which are not normally found in large numbers in clean soil and water.

The only guarantee that drinking water is safe for human consumption is achieved with the addition of

High Test Calcium Hypochlorite (HTH) as recommended in the Manual of Naval Preventive Medicine, NAVMED P-5010, Chapter 6. Thorough distribution of HTH throughout the body of water being treated must be accomplished to ensure disinfection. This can best be accomplished by adding the chlorine during the filling operation. The supernate from the pre-mixed HTH should be poured into each tank via the sounding tubes as necessary.

It is difficult to determine the exact amount of chlorine compound required to achieve a desired residual. The physical quality of the water and the presence of bacterial contamination serve to dissipate chlorine residual levels. If the chlorine residual is read immediately following chlorination, the reading will be false; however, if a chlorine reading is performed immediately following a 30-minute contact time, it will be accurate and dependable. A mere five-minute delay here could again interfere with accurate readings. The parts per million (ppm) interpretation obtained by color comparison will determine the level of FAC (Free Available Chlorine) that is present in your water supply. This level may fall short of expectations. If the reading is zero, then re-chlorination followed by another 30-minute wait is required. The chlorine residual indicated on your color comparator now represents the true amount of chlorine available to kill pathogenic (disease causing) bacteria and acts as a safeguard against possible contamination. Daily follow-up chlorine residual checks should be made throughout the ship to ensure that the desired level is being maintained.

RABIES PREVENTION

Editorial by Hildreth, E.A., Ann Intern Med 64(6): 1357-1360, June 1966.

During the past decade new principles have evolved concerning post-exposure prevention of rabies. The use of vaccine therapy and hyperimmune serum therapy have been widely discussed. The latest information on this type of therapy has been published by the World Health Organization (WHO) Expert Committee on Rabies in the *WHO Technical Report Series* (No. 321, 1966).

One area of rabies prevention that needs to be re-emphasized is prompt local wound care. All evidence points to the necessity of cleansing the wound thoroughly *at once*.

Good local care of wounds contaminated with rabies virus appears to decrease significantly the development of rabies. The wound should be thoroughly cleaned with a 20% soap solution throughout its entirety. It is necessary to remove all blood and organic debris because of their neutralizing action on many disinfectants. Soap and anionic detergents neutralize quaternary ammonium compounds and therefore must be carefully rinsed from the wound area before viricidal agents of this type are used. The best rinsing fluid is water.

After careful rinsing, 1:500 or 1:1,000 dilutions of benzalkonium chloride (FSN 6505-149-8705) or cetrimonium bromide can be used for careful, thorough irrigation of the wound. These concentrations of benzalkonium and cetrimonium are not harmful to the tissues. A 1:10,000 dilution of iodine is viricidal for rabies virus and is acceptable as a replacement for the quaternary ammonium compounds noted above. Other substances that are viricidal for rabies virus, and can be used, are 70% ethanol and tincture of thimerosal.

On occasion a patient will present himself or treatment long after rabies exposure. Regardless of how many days have elapsed since the bite, as long as the patient is not overtly ill with rabies, the wound should still be thoroughly cleaned.

After the wound has been cleaned, vaccine and serum therapy should be instituted as directed in the WHO publication.*

Cleansing the wound alone, without vaccine and serum, will not assure prevention of rabies. Proper care of the wound, however, can increase the effectiveness of the preventive therapy significantly.

Although it is a quite simple procedure, cleansing

of the wound is many times done hastily and superficially, in the rush to institute vaccine and serum therapy.

TUBERCULOSIS AND ICTERIC HEPATITIS

J. Langer, K. Czyzewski, J. Mieszkowski, W. Palka, Zakopane, Poland, Dis Chest 50(1): 33, July 1, 1966.

A study of 359 cases of pulmonary tuberculosis showing icteric hepatitis in the course of treatment is presented. It seems that jaundice has a favorable action on the course of pulmonary tuberculosis. Biliary salts seem to be the main agents in this process through their anti-inflammatory, cortisone-like and chemical properties which may exert a nonspecific desensitizing action. Decrease of positiveness in tuberculin tests noticed in connection with the improvement of the pulmonary condition shows that there is no reduction of immunologic potentials in pulmonary tuberculosis patients with icteric hepatitis.

CONGENITAL MALARIA

USDHEW PHS CDC, Morb & Mort Wkly Rpt 15(34): 289-290, Aug 27, 1966.

A case of congenital malaria in a 2½ month old female infant, born to Philippine parents, was detected in Chicago, Illinois. The infant born there after a full-term pregnancy and normal delivery, was in good health until the onset of spiking fever at 72-hour intervals at the age of 7 weeks. *Plasmodium malaria* schizonts were found in blood smears.

The mother had no history of malaria either in the Philippines or since arrival in the United States in 1963. Careful review, however, of thin blood smears obtained from her during a routine prenatal visit and at the time of delivery revealed one trophozoite and 6 trophozoites, respectively.

Neither mother nor child had a history of transfusions. The diagnosis of *P. malariae* infection in both cases was confirmed by fluorescent antibody studies. Both patients were treated with anti-malarial chemotherapy.

The father had a history of malaria during childhood in World War II in the Philippines, but has had no attacks since that time. Repeated blood films have been negative for malaria organisms.

* See U. S. Navy Medical News Letter 47: 13-15, (10 June) 1966—
"Rabies in Man and Animals."

Edit. Congenital malaria is a rare occurrence even in highly endemic areas in the world. The incidence estimates vary from 0.03 to 9.6%. Spitz (Bull Wld Hlth Org 21: 242, 1959) observed no malaria parasites in blood films of 137 newborn

infants, although the maternal side of the placentae in these cases were heavily infested.

The above case report is of special interest in that the mother had no clinical history of malaria but apparently had a persistent low parasitemia.

KNOW YOUR WORLD

Did You Know?

That a total of 2,703 cases of encephalitis were reported in the United States in 1965?

Epidemiologic follow-up of cases and laboratory tests on blood specimens, showed that for 297 cases, the etiologic agents were arthropod-borne viruses—Eastern, Western, St. Louis and California. In 981 cases, encephalitis was a complication or sequela of mumps, measles (rubeola), chickenpox, rubella and other infections. For almost half of the cases (1,425), the etiologic agent or cause still remains unknown. (1)

That there would be 12 million fewer cases of chronic illness if all Americans were nonsmokers?

According to a recent survey, smoking is related to the existence of 300,000 coronary cases, 1 million cases of chronic bronchitis or emphysema, nearly 2 million cases of sinusitis, and more than 1 million cases of peptic ulcers. Three million man-days of restricted activity and 900,000 days spent ill in bed are part of the price of smoking according to the survey. The Social Security Administration pays more than \$60 million a year to persons disabled by emphysema. (2)

That the oyster shells that are exposed over a large portion of southern San Francisco Bay have been used since 1924 as the basic raw material for cement manufacture? (3)

That it will cost \$6,500,000 to control water pollution at only 25 military establishments scattered throughout the United States?

The military construction appropriations bill for fiscal year 1967 sets this amount as a start towards the *Defense Department's* portion of *cleaning up*

pollution caused by Federal facilities. This type of appropriation is bound to become a much larger budget item in the future for the three Services. (4)

That 15 tons of uncooked bacon sealed in metal cans were sterilized by penetrating rays that destroyed bacterial and other microorganisms?

This was the first large-scale field test of the effectiveness of gamma radiation for sterilizing foods. (5)

That firearm accidents will probably toll about 2,200 lives in 1966, or a death rate of 1.1 per 100,000 population?

About 19 million persons were issued hunting licenses in 1964 according to the Fish and Wildlife Service of the U. S. Department of Interior. Firearm safety program needs to be broadened and intensified not only among hunters but in members of their households and all who possess firearms. (6)

That an epidemic of malaria is reported to have killed some 2,000 persons in 1 month on Marajó, the largest island in the Amazon River delta?

The island's supplies of drugs were apparently exhausted early in the epidemic. Brazilian public health officials sent 2 physicians to distribute anti-malarials to 300 people. The death toll in the largest city of the island has been estimated at 1,200. All 400 inhabitants of one village were dead and unburied in their shacks. (7)

References

1. USDHEW PHS CDC Morb & Mort Annual Supplement, Summary 1965, 14(53): 1-60 Oct 14, 1966.
2. Wisc State Bd of Hlth, Qtrly Bull "Health", 17(8): 28, Fall 1966.
3. Sci News 90(3): 43, July 16, 1966.
4. Water Info Center, Inc, Water Newsltr 8(21): 2, Nov 10, 1966.
5. Sci News 90(16): 313, Oct 15, 1966.
6. Metropolitan Life Ins Co Stat Bull 47: 1-3, Sept 1966.
7. Medical World News 7(41): 35, Nov 4, 1966.

EDITOR'S SECTION

AMERICAN BOARD OF OB-GYN

Applications to take Part II (oral) examination November 6–10, 1967 will be accepted in the office of the Secretary during January and February 1967—postmarked no later than February 28th. Applications must be accompanied by duplicate lists of patients dismissed from candidates' service during the twelve months immediately preceding the month of application. A sample format to be followed in the listing of patients is enclosed in each application form.

ALL APPLICANTS MUST HAVE PREVIOUSLY PASSED THE PART I (WRITTEN) EXAMINATION.

Application forms and Bulletins may be obtained by writing to the office of the Secretary, Clyde L. Randall MD, 100 Meadow Road, Buffalo, New York 14216. Prospective candidates are urged to review the current Bulletin of the Board for complete information on the requirements for application.

Diplomates and candidates are requested to keep the Board office advised of their current address.—Training Administrative, BuMed.

SPACE AND ASTRONAUTICS ORIENTATION COURSE

This course has been established to give senior officers of the Navy a better understanding of this new technology, its application to naval warfare, and its important role in national defense. The course is in consonance with the Navy's global mission and emphasizes the significant impact of astronautics on seapower. It is primarily designed for those senior officers who have not had the opportunity to gain knowledge of astronautics and current space programs. A highlight of the course is a visit to the space vehicle launch and control facilities at Point Arguello Naval Missile Facility and Vandenberg Air Force Base.

Location: Naval Missile Center, Point Mugu, California

Duration of Course: Four days (Tuesday-Friday)

Convening Dates of Course:

24 January	1967	11 July	1967
14 February	1967	12 September	1967
14 March	1967	24 October	1967
25 April	1967	14 November	1967
9 May	1967	5 December	1967
6 June	1967		

BUMED Quota: One for each class

Deadline Date to Apply: Immediately for the 24 January and 14 February courses, and six weeks in advance for the remaining courses.

Eligibility: Rank of Lieutenant Commander and above. TOP SECRET Security Clearance required.

In view of the shortage of travel funds for Fiscal Year 1967, only a limited number of officers can be authorized to attend these courses on travel and per diem orders chargeable against Bureau of Medicine and Surgery funds. Eligible and interested officers who cannot be provided with travel orders to attend at Navy expense may be issued Authorization Orders by their Commanding Officers following confirmation by this Bureau that space is available in each case. Requests should be forwarded in accordance with BUMED INSTRUCTION 1520.8 Series and comply with the deadline dates indicated above. All requests must indicate that a security clearance of TOP SECRET has been granted to the officer requesting attendance, and if Bachelor Officer's Quarters are desired.—Training Branch, BuMed.

STOKES STRETCHER

A rigid Stokes-type litter constructed of plastic with a flotation capability is currently being developed. It is expected that this will be field tested and evaluated at the Naval Medical Field Research Laboratory at Camp Lejeune, North Carolina, in late 1966 or early 1967. With a flotation capability, the safety of patients will be increased, especially at sea when being transferred between ships. The plastic litter as designed should be as durable as its metal counterpart and will be extremely resistant to corrosive environments.—Public Affairs Office, BuMed.

SURGERY AT SEA

NOW

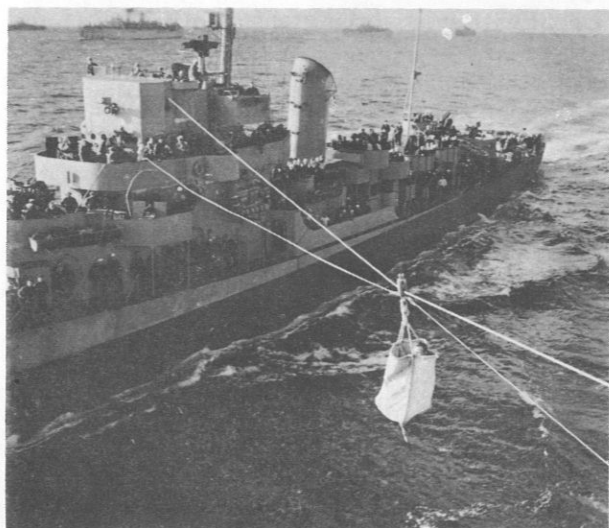


LT C. L. Chambers examining ENS Tom Stevens on the guided missile destroyer USS Towers, deep in Tonkin Gulf, September 1966. Symptoms, physical findings, and results of laboratory determination were those of acute appendicitis. Less than two hours



later, the patient was being transferred by helicopter to the aircraft carrier USS Kearsage where his appendix was removed. Diagnosis: acute appendicitis. Result: Uneventful Recovery.

THEN



Medical officers, surgical operating room technician, and surgical operating room equipment transferred from USS Hermitage to USS Mills, destroyer escort, by coaling bag and then to SS Santa Elisa, merchant marine cargo vessel, by boatswain's chair, October 1944, to operate on a member of the gun crew in the latter. Probable diagnosis of appendicitis made after evaluation of patient's symptoms, and physical signs described by LT(jg) J. I. McFadden commanding officer of the gun crew by radio telephone (no medical officer or Pharmacist's Mate aboard the cargo vessel). Operation was performed

in the ward room. Diagnosis: appendicitis with perforation of the appendix and localized peritonitis. Result: uneventful recovery. Except for one medical officer, who was left in the SS Santa Elisa to manage the postoperative care of the patient, the surgical team returned to the USS Hermitage by boatswain's chair and coaling bag, this time via the USS Finch. Photographs are of medical officer being transferred in coaling bag and of CDR John Ohanneson, Senior Medical Officer, USS Hermitage, in boatswain's chair.

BGEN J. M. BLUMBERG PROMOTED TO MAJOR GENERAL

Brigadier General Joe M. Blumberg MC USA, The Director, Armed Forces Institute of Pathology, was promoted to Major General during his recent staff and consultative tour through Europe and the Far East. The second star was pinned on his shoulder by LGEN James W. Wilson, Commander of the Thirteenth Air Force, at Clark Air Base, Manila, Republic of the Philippines. From Manila General Blumberg flew across the International Dateline and landed in Honolulu, Hawaii on November 11, where MGEN Byron L. Steger MC USA, Chief Surgeon of the U. S. Army in the Pacific, temporarily demoted General Blumberg by removing the newly-gained star and then pinned it on again promoting him for the second time that day.

Now the highest ranking pathologist in the U.S. Armed Forces, General Blumberg has been Director of the AFIP for the past three years. AFIP is the central pathology laboratory for the Army, Navy,



Air Force, Veterans Administration, and Public Health Service.

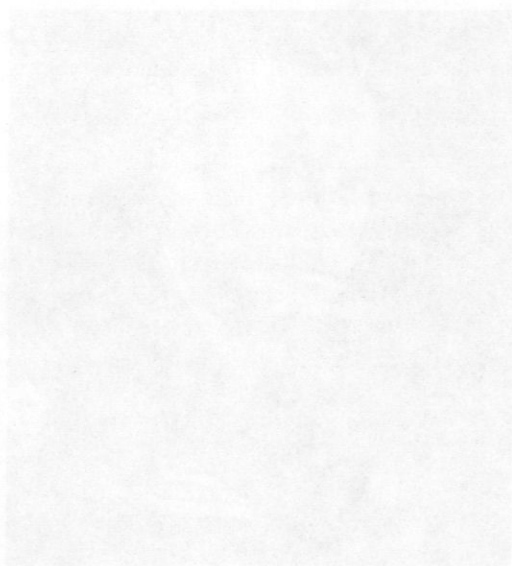
General Blumberg is married to the former Catherine Weller of Washington, D.C., who is also a medical doctor. They reside in Washington at 5007 Jamestown Road.—AFIP, Washington, D.C.

DEPARTMENT OF THE NAVY

BUREAU OF MEDICINE AND SURGERY
WASHINGTON, D.C. 20390

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BOB L. ALBRIGHT, PROMOTED
TO MAJOR GENERAL

Major General L. Albright, USA, was promoted to Major General during the ceremony held at the Pentagon on May 1, 1968. The ceremony was presided over by the Secretary of Defense, Mr. Robert McNamara. Major General Albright is currently assigned to the position of Deputy Chief of Staff for the Surgeon General's Office, Department of the Navy. He is a graduate of the United States Military Academy at West Point, New York, and has served in various capacities in the Army and Navy. Major General Albright is a member of the American Medical Association and the American College of Surgeons. He is also a member of the National Academy of Sciences. Major General Albright is married and has three children.

Albright, L. (Bob). 1917-1968. Surgeon General's Office, Department of the Navy. Major General. Promoted to Major General during the ceremony held at the Pentagon on May 1, 1968. The ceremony was presided over by the Secretary of Defense, Mr. Robert McNamara. Major General Albright is currently assigned to the position of Deputy Chief of Staff for the Surgeon General's Office, Department of the Navy. He is a graduate of the United States Military Academy at West Point, New York, and has served in various capacities in the Army and Navy. Major General Albright is a member of the American Medical Association and the American College of Surgeons. He is also a member of the National Academy of Sciences. Major General Albright is married and has three children.